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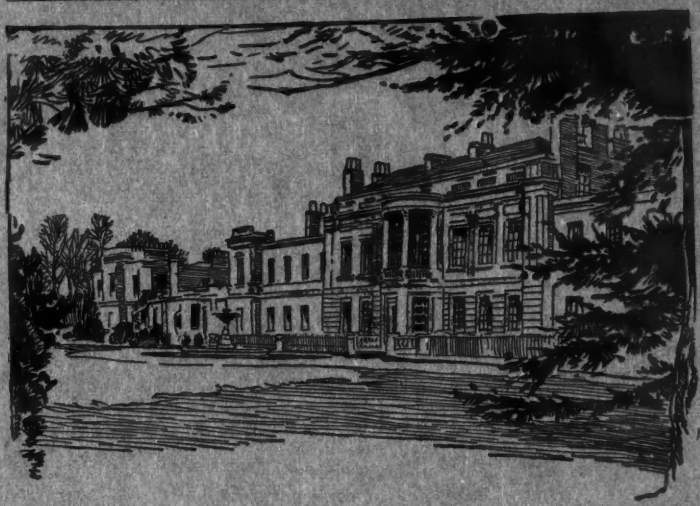

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The Bristol Medico-Chirurgical Journal

" Scire est nescire, nisi id me Scire alius sciret."

AUTUMN, 1948.

BRONCHIAL CARCINOMA

BY

W. W. D. THOMSON, D.L., M.D., B.Sc., F.R.C.P.*

Professor of Medicine, The Queen's University of Belfast.

THE first definite clinical description of cancer of the lung I can discover is given by Boerhaave (Zimmerman, 1782) with such dramatic skill that I cannot refrain from quoting some extracts therefrom. The Marquis of St. Auban was a strong and active man until a continual cough and persistent pain in his chest began to mar his previous splendid health. His breathing became difficult, he usually sat upon his bed, leaning forward with his elbows on his thighs. He could get no sleep night or day. In despair his physician sought the services of the great Dutch physician who put aside the tentative diagnosis of abscess of the lung owing to lack of constitutional symptoms, and inclined to the opinion that undue resistance of the thoracic walls caused the pain, dyspnœa and sense of suffocation. Ten months after the onset of symptoms the Marquis died. Boerhaave obtained permission to open the body which was, despite the victim's suffering, by no means emaciated. The whole left cavity of the chest was filled with a white substance, which had invaded the heart and compressed the bronchi. Boerhaave was stupefied at the sight of this singular phenomenon which he regarded as a steatoma and described the condition under the title " *Atrox Rarissimusque Morbus.*"

*From an address delivered to the Bristol Medico-Chirurgical Society on February 18th, 1948.

In 1912 Adler published his classical work, *Primary Malignant Growths of the Lungs and Bronchi*, and excited a fresh interest in the subject. Since then the great developments in radiology and bronchoscopy, in anæsthetics and thoracic surgery have given sufferers from this terrible and common disease a ray of hope, provided an early diagnosis be made. The urgency and importance of the problem is shown by the Registrar-General's latest statistical review, where we find that primary cancer of the lung in the male is exceeded in frequency only by cancer of the stomach, rectum and colon : and that 14 per cent. of men who die of cancer in the prime of life, between the ages of thirty-five and fifty-five, die from pulmonary carcinoma.

From the combined observations made by clinicians, bronchoscopists, radiologists and pathologists a fairly complete picture of cancer of the bronchus and its modes of behaviour can be obtained. The great majority of these growths have their origin in the main bronchus 1 or 2 cm. below the bifurcation of the trachea ; less frequently in bronchi of the second order. This is the hilar or central type. On the other hand the tumour may develop from one of the smaller bronchi producing a circumscribed tumour mass at a distance from the hilum—the peripheral or parenchymatous type. Kramer and Som (1936) have described the bronchoscopic appearance of a very early lesion in a patient who presented few pulmonary signs and in whom X-ray examination of the chest revealed no abnormality. The normal bronchial marking was distorted over a small area one centimetre in diameter where the rings seemed to be drawn close together and over which the mucosa presented a ruffled and wrinkled appearance. Biopsy of the overlying mucosa revealed an infiltrating squamous-celled carcinoma. From such a lesion growth may proceed by submucous infiltration around the bronchus. As growth proceeds more of the bronchial circumference is involved until a tubular stenosis is produced. On the other hand the growth may early break through the mucosa and protrude into the lumen appearing as a granular, friable and sessile tumour which quickly produces obstruction. Or again the primary growth, passing the cartilaginous barrier of the bronchial wall, may invade the lung tissue, being demonstrated on X-ray examination as a shadow at the hilum. These early stages are but passing phases, for soon occlusion of the bronchus follows and collapse ensues. Once collapse takes place the disease progresses rapidly.

Clinical History.—Let us pause to consider the possibility of diagnosis at this comparatively early stage, for in no malignant condition is early diagnosis more important. The great difficulty in early diagnosis lies in the paucity of symptoms. These are so unobtrusive that the patient does not seek medical advice, so commonplace that the physician does not suspect serious trouble.

Cough is the first symptom to appear : the onset of an unexplained cough should excite the suspicion of cancer of the lung when this symptom occurs in a patient of middle age hitherto free from respiratory ailments. Often the cough differs in no way from that found in ordinary catarrhal conditions of the bronchial tract. "Short", "dry", "something sticking that I cannot get up", are common descriptions. Its persistence and failure to respond to treatment are characteristic. Later, as the obstruction increases, the cough is apt to become paroxysmal, and at times resembles whooping-cough. An "asthmatoid wheeze" may attract the patient's attention (Jackson, 1930). The sputum at this stage, if present at all, is generally scanty and mucoid. If the sputum becomes streaked with blood the patient is fortunate, for medical advice will probably be sought. When streaking occurs it is apt to persist for days, but in this series hæmoptysis was recorded in less than 40 per cent. of the cases. In such an event pulmonary tuberculosis is generally considered, and the repeated failure to demonstrate tubercle bacilli and the absence of direct radiological evidence of disease in the chest, instead of exciting suspicion, is apt to engender a false feeling of security. The possibility of an early growth demonstrable by the bronchoscope is overlooked. Dyspnœa on exertion may be a comparatively early symptom. To sum up, the association of persistent cough, scanty sputum (especially if bloodstained) and respiratory discomfort should always in a male patient of thirty-five or over raise the suspicion of cancer of the lung and lead to a thorough investigation.

In the earlier stages of obstruction the lumen is simply narrowed : air can pass in and out, but in diminished amount. The "asthmatoid wheeze" can sometimes be found if listened for at the open mouth of the patient : this sound is rarely audible at the chest wall.

Later, obstruction has increased so that, though during inspiration dilatation of the invaded bronchus is sufficient to allow air to enter, on expiration it cannot escape. The result will be an emphysema in the corresponding lobe. It requires an expert to demonstrate convincingly the physical signs, and in the early stage clinical examination may yield completely negative results. But the suspicions roused by the patient's story must not be disregarded. Any sputum obtainable should be examined by the technique of Dudgeon and Wrigley (1935), who by means of a wet-film method were able to demonstrate particles of malignant growth. Out of 110 cases of primary cancer of the lung, diagnosis was made from the type of cell found in the sputum in sixty-eight.

X-rays.—In the very early stage no direct evidence of growth may be seen in plain X-ray film, but lipiodol may be of the greatest service. When the tumour grows into the lumen it produces a bronchogram showing a filling defect. The infiltrative type of growth

produces a concentric filling defect with irregular borders. Bronchography is also of value in that it may reveal filling defects beyond the range of vision of the bronchoscope.

It has been mentioned that not infrequently the growth from the main bronchus may infiltrate the lung parenchyma before producing broncho-stenosis. In such an event X-ray examination discloses a shadow situated at the hilum. The border of this shadow may be well defined or may be ragged with strands of growth passing outwards along the branches of the bronchus. A lateral view should always be taken when such a hilar shadow is seen lest this may be a pseudo-hilar one. It is in the diagnosis of the hilar shadow that most radiological errors are made.

Bronchoscopy.—The bronchoscope offers the most decisive method of diagnosis, and may reveal a lesion too small to be demonstrated by any other method. Kramer and Som (1936) carried out a bronchoscopic examination in 300 cases of pulmonary carcinoma, and in 74 per cent. the diagnosis was confirmed by histological examination of biopsy tissue so obtained. By a combination of clinical, laboratory, radiological and bronchoscopic methods it is evident that, if suspected and sought for, many bronchial carcinomata situated in the neighbourhood of the hilum can be diagnosed in a relatively early stage.

Late Stage.—With the development of complete stenosis the disease enters on its terminal stage. The radiological signs of collapse appear, and the area supplied by the occluded bronchus is seen as an opaque shadow. The boundaries of the opaque area are concave (if viewed in a suitable projection) and the adjoining lobe or lobes have become emphysematous. In many cases at the site of the stenosis a rounded or irregular shadow fusing with the opaque area can be seen which represents the expanding tumour. The collapsed portion, its airless alveoli filled with exudate, forms a suitable nidus for inflammatory changes: hence pneumonic consolidation and abscess formation are frequent complications. The retention of septic material in the blocked bronchi and alterations in pleural pressure favour the progress of bronchiectasis. The pleura becomes involved in practically every case, an effusion may develop, adhesions may be produced or plaques of new growth stud the membrane. The growth may necrose and cavity formation occur. Hence many patients date the onset of their illness from symptoms referable to the pulmonary complications which follow in the wake of collapse. This was the most frequent type of onset found in this series, comprising 40 per cent. Chills, colds in the chest, "influenza", "pneumonia", pleural effusions with or without previous pain in the chest, were common clinical stories. It is surprising that the spitting of blood was recorded in only 37 per cent.

The persistence of the bleeding is a striking feature. The sputum often continues to be stained for weeks.

The usual clinical features found in this group are dullness on percussion with loss of breath sounds at one base. Often all signs of a pleural effusion are present, but the exploring needle fails to find fluid and may give the impression of entering a solid resisting mass. Occasionally the heart sounds can be heard distinctly over this dull area. The exploring needle passing through a pleural cavity, obliterated by adhesions, may enter an abscess or bronchiectatic cavity and withdrawing pus lead to an error in diagnosis. Or pleurisy with effusion, serous, hæmorrhagic or purulent, may be found. A latent and symptomless pleural effusion, especially if hæmorrhagic, is almost pathognomonic of bronchial carcinoma. Even when a pleural effusion masks the lesion stenosis of a bronchus can easily be demonstrated by tomography. When purulent fluid is withdrawn the diagnosis of empyema is a natural one. Operation was performed in nine cases in this series in which empyema or lung abscess had been diagnosed.

Pain occurred in 65 per cent. of my patients during some stage of their illness. In 15 per cent. it was the symptom for which they first sought medical advice. Pain is not a symptom in the early stage. In the hilar type pain is generally related to pleural involvement. Still later in the illness pain may be due to nerve compression or erosion of the bony framework of the thorax.

In 11 per cent. of this series the growth involved the bronchial glands to a greater extent than the lung : consequently pressure on structures in the mediastinum evoked the presenting symptom. In the course of the disease (though often late) nearly 50 per cent. develop pressure signs. The orthopnœa so often witnessed as a terminal event is found associated with mediastinal pressure narrowing the great air passages. Difficulty in breathing may at times simulate attacks of asthma.

A very important group causing great difficulty in diagnosis is the type whose initial symptoms are referable, not to the primary growth in the chest, but to metastases elsewhere. These constituted 10 per cent. of the series. Metastatic deposits in the brain arising from a latent cancer in the lung have been so often diagnosed as primary cerebral tumours that a routine X-ray of the chest should be carried out to avoid this source of error. The diagnosis is scarcely less puzzling when the presenting symptoms are caused by secondary deposits in bone. Metastases in the supraclavicular or axillary glands may be helpful in diagnosis. In one exceptional case an axillary gland became enlarged more than a year before any other evidence of the disease was present : but as a rule pulmonary growth is advanced before the superficial glands become palpable.

Symptoms which led to diagnosis in author's 130 cases may be summed as follows:

	Per cent.
Early: cough, sputum (sometimes bloodstained), changes in respiration	17
Pulmonary complications following occlusion of bronchus	40
Pain	15
Pressure symptoms	11
Metastases	10
Others	6

Other types of primary cancer of the lung are less frequently found. Of special importance are the tumours which are found separate from the hilum in the parenchyma of the lung. They arise from small bronchi. They are generally circumscribed and produce spherical, well demarcated growths in the lung. This peripheral type of tumour may remain latent for long periods. They are notoriously prone to give rise to early metastases in distant organs which may be the first indication of their presence. Mass radiography may demonstrate their unsuspected presence. The solitary rounded shadow found may be difficult to distinguish from an encysted interiobar empyema, from a hydatid or other cyst or from a benign neoplasm.

Among the Belfast series of cases a type in which the pleura was mainly involved was not uncommon, five cases being of this nature. Two of these were traced to a small adenocarcinoma originating in one of the lesser bronchi. In one of these an extension of the growth from the pleural mass invaded the vertebral column causing paraplegia. In three cases, however, the tumours were shown to be mixed-celled sarcomata. In each the growth was confined to one pleural sac and the bronchi were unaffected. The presenting symptom was pain which was accompanied by pleural friction and effusion.

Differential Diagnosis.—The difficulties introduced into the diagnosis of primary cancer of the lung by the pulmonary complications inherent in the disease or by metastases elsewhere in the body have been considered. Careful repeated bacteriological examination of the sputum and X-ray examination should exclude pulmonary tuberculosis. It must be remembered, however, that error is possible in the reverse direction, as the presence of tubercle bacilli in the sputum does not necessarily exclude cancer of the lung, the two conditions being occasionally co-existent.

The diagnosis between fibrosis and carcinoma of the lung is often very difficult. The patient's preceding history may assist, lipiodol injection will generally show patent bronchi and bronchiectatic

cavities. Bronchoscopy will probably reveal a ragged granulomatous condition of the bronchi and quantities of pus may be seen exuding from their orifices.

In 6 per cent. of this series evidence of previous syphilis was obtained. Syphilitic sequelæ can closely simulate bronchial carcinoma. The differential diagnosis between aneurysm of the aorta and intrathoracic neoplasm is discussed in every text-book of medicine. In one case under my care the diagnosis was only settled by surgical exploration.

When Hodgkin's disease chiefly affects the mediastinal glands the simulation of primary cancer of the lung may be very close. Secondary growths in the lung from a latent primary focus may cause compression of a bronchus.

Considerable attention has been drawn in recent years to the question of benign tumours of the bronchi. By far the most important is the bronchial adenoma which generally arises in the primary or secondary bronchi. Their histological structure often suggests a malignant growth, but from their long clinical course and absence of metastases they are regarded clinically as benign. There are two distinct groups of symptoms—those due to the adenoma—cough and recurrent hæmoptysis, and those due to obstruction causing the secondary features of collapse and infection of the lung. Treatment is directed to prevent these sequelæ, and the prognosis is good only when diagnosis, which depends on bronchoscopy, is made early and followed by treatment.

REFERENCES

Zimmerman (1782). *A Treatise on Experience.* London. Book iii, p. 232.

Adler, I. (1912). *Primary Malignant Growths of the Lungs and Bronchi.* New York.

Kramer, R., and Som, M. L. (1936). *Arch. Otolaryng.*, 23, 526.

Jackson, C. (1930). *J.A.M.A.*, 95, 640.

Dudgeon, L. S., and Wrigley, C. H. (1935). *J. Laryng. and Otol.*, 50, 752.

SOME POINTS IN THE TREATMENT OF DIABETES

BY

G. A. SMART, M.D., B.Sc., M.R.C.P.

Bristol.

Diabetic Coma. Patients who die of diabetes mellitus die in diabetic coma. This highly dangerous complication is preventable, therefore the first aim in treatment should be to prevent it. Although the cause of coma in diabetes is not known, the condition is closely correlated with ketosis, which almost invariably precedes its onset. The ketotic or pre-comatose state need never be fatal: whereas, however good the treatment, cases of true coma often die. Moreover, diabetic coma is rarely of acute onset; at least twenty-four hours (and usually several days), elapse during which gross ketosis develops before the onset of loss of consciousness and circulatory collapse. The best insurance policy therefore in the prevention of diabetic coma is the regular testing of the urine for ketone bodies. This is of much greater importance than testing for sugar. It cannot be over-emphasized that hyperglycaemia itself is not in the short term harmful: safety from diabetic coma lies in ensuring freedom from ketosis and not in ensuring normal blood-sugar.

Ketosis arises whenever carbohydrate metabolism is abnormally low, either from the inadequate intake of carbohydrate or from insufficient or inefficient action of insulin or both. Ketosis is therefore treated by giving more insulin or by ensuring a greater carbohydrate intake or both.

It is essential that the patient be correctly instructed in this matter. Most patients have the firmly rooted idea that *sugar* in diabetes is harmful. If they test the urine for this substance only they may, in the pre-comatose state, note an excessive glycosuria, cut down their carbohydrate, and thus precipitate the onset of true and dangerous coma. I emphasize this point, because I am constantly having to treat patients in diabetic coma precipitated in this manner. If the patient is only capable of carrying out one kind of test on the urine let it be for ketones.

The classical tests for ketones (ferric chloride or Rothera's), are not suitable for patients.

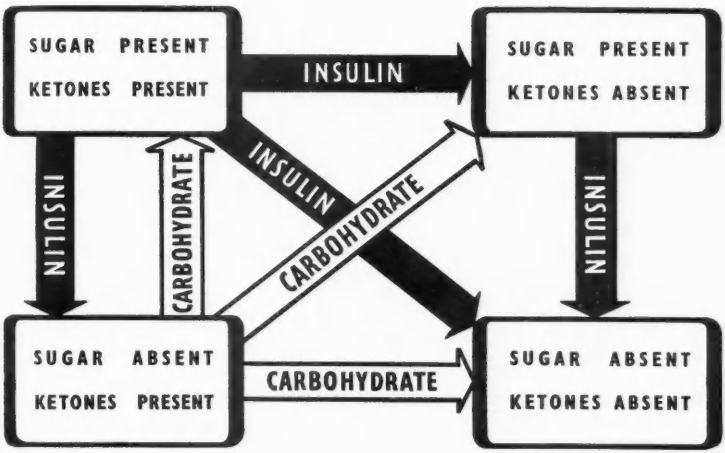
Powder for Ketone Test.[1]

℞ Sodium nitro prusside	0.5 per cent.
Anhydrous tribasic sodium phosphate	12.5 per cent.
Ammonium sulphate	87.0 per cent.

Keep the powder dry in a well-corked bottle.

In a very simple test a small pile of powder is moistened with the urine and the colour read at the end of five minutes : this is well within the capacity of every patient. The test is very sensitive and instructions must be given that there is danger only when the purple colour is very deep, although with proper treatment this should never occur.

If the patient has not got a low renal threshold in addition to diabetes (and this combination is not unknown), and if the urine is tested both for sugar and ketones a simple scheme of treatment can be adopted.



The figure shows, in simplified form, four main types of results which may be obtained when the urine is tested. If ketones are present and sugar is absent, then carbohydrate metabolism is insufficient : the remedy is to increase the carbohydrate in the diet. If sugar is present with or without ketones more insulin should be given. Diabetics taking insulin are better with a slight glycosuria (shown by Benedict's test), since they are then less liable to have hypoglycaemic attacks. About 50 per cent. of diabetic patients are capable of understanding this scheme : it enables them to become

self-reliant and to alter their dosage of insulin when this becomes necessary without unduly frequent reference to their medical adviser. The most important advice to those who cannot follow the scheme is that they must test the urine every morning for ketones and report at once if the result is positive.

Insulin or Restricted Diet? This focusing of attention on ketonuria rather than on glycosuria is necessary whether the diabetic is treated with insulin or not. Ketosis is always a danger signal and active steps should be taken to combat it. Nevertheless, many diabetics do not exhibit ketosis when first seen, the glycosuria apparently producing the main symptoms. Greater improvement may be obtained in such patients by a restricted diet and by measures to increase the utilisation of carbohydrate than by small doses of insulin. Incongruous as it may seem, when such cases are treated with insulin they frequently require larger doses than those who, without insulin, would rapidly develop ketosis and die. The requirement of insulin for stabilisation has no correlation with the severity of the diabetes.

Diabetes mellitus is in all probability not a single disease, but a syndrome including at least two quite separate conditions. It is well recognized clinically that there are two main types of diabetes : the thin patient, responsive to relatively small doses of insulin, becoming ketotic easily and liable to have rapid fluctuations in the blood-sugar levels ; and the obese patient, rarely ketotic, in whom large doses of insulin are required to lower the blood sugar. These two types have been recognized on anthropometric grounds[2] and may correspond to the two experimental types of diabetic animal, those who have suffered destruction or removal of the pancreatic islets on the one hand, and on the other those receiving large doses of anterior pituitary extract. In general, underweight diabetics require insulin and those who are overweight do well with suitable dietetic treatment : but patients showing ketosis, young diabetics, and those with complications such as retinopathy, infections or gangrene, should always receive insulin. In diabetes of recent onset treatment should include insulin, be urgent, rigorous and thorough. The reason for this is the new hope, derived from animal experiments, that sufficiently early and thorough treatment may cure the diabetic state.

Prevention of Diabetic Gangrene. In the older diabetic the most serious complication, apart from coma, is gangrene. This occurs almost invariably in the feet, danger points being toe-nails and callosities. There is no unequivocal evidence concerning the effect of various diets or of a high blood sugar on the development of arteriosclerosis in diabetes, but what evidence there is suggests that a diet high in carbohydrate is least likely to hasten its onset. Lawrence[3] has pointed out that patients with haemo-chromatosis

are not especially liable to arteriosclerosis. Possibly poor control of diabetes contributes towards the development of arteriosclerosis, or perhaps both diabetes and arteriosclerosis are due to the same underlying causes. At all events, in the present state of our knowledge, it is wise to regard any irregularity in the control of diabetes as contributory towards the advance of arteriosclerosis. Diabetic gangrene may also in part be due to the presence of peripheral neuritis should this involve autonomic fibres controlling peripheral blood-flow.

To lessen the risks of gangrene developing, the diabetes should be strictly controlled, the feet should be kept perfectly clean, shoes should be chosen for fit, not fashion, and regular attendance should be made on the chiropodist. Injuries to the feet even of the most minor character should be avoided, and should be carefully treated if they do occur. Particular warning should be given to patients that they should never use uncovered hot-water bottles, since if peripheral neuritis be present pain-sensation may be diminished with a consequent risk of burns.

Dietetic Requirements. It is probable that the diabetic requires a diet containing more of the vitamin B complex than a normal person. Many members of the vitamin B complex act as co-enzymes in carbohydrate metabolism and evidence of deficiency is found not infrequently in diabetics, even when the diet contains amounts of vitamin B complex considered to be adequate for the normal individual. Angular stomatitis, sometimes accompanied by superficial glossitis, is quite common among diabetic patients ; it is very difficult to treat and often does not respond to large amounts of the B group of vitamins. It is usually considered to be the result of riboflavin deficiency, but this is undoubtedly an over-simplification. The lesion may develop or grow worse shortly after the start of insulin therapy. Sydenstricker (1939)[4] reports two such cases suggesting that the deficiency is made apparent by the rapid increase in carbohydrate metabolism brought about by the insulin. True pellagrous skin lesions are sometimes seen, but these usually respond satisfactorily to large doses of nicotinamide. A B-complex deficiency may play a part in the peripheral neuritis so common in diabetics, and cases have been reported in which the peripheral neuritis has developed rapidly after the start of insulin therapy. In general, however, peripheral neuritis is more common among diabetics who have had inadequate therapy over long periods.

It goes without saying that, in poorly controlled diabetes, when amino-acids are being converted in large quantities into carbohydrate there is likely to be some upset in protein metabolism. Rarely cases are seen in which there is a generalized oedema associated with low plasma-protein levels, but usually there is no gross deviation from the normal. It may be that the proportions of constituent plasma

proteins may be upset, although the total level is normal. This has been found to be particularly marked in subjects with diabetic retinopathy and it was alleged that, where normal proportions could be obtained by feeding high protein diets, the retinopathy showed a remarkable improvement. There is sufficient evidence that diabetics should ensure that their diet is rich in vitamins of the B complex and that it contains adequate amounts of first-class protein.

The diet of diabetics receiving insulin should be as nearly normal as possible, but it should contain a larger proportion of the protective foods than usual. There has been much controversy over the relative values of high-fat and high-carbohydrate diets, equally good results being claimed by both schools. Diabetics taking insulin and feeling well frequently do not keep very carefully to a strictly prescribed diet. Provided insulin is used there seems to be no virtue in restricting the patient from eating just what he likes—including sugar in tea, if desired! It is alleged that more insulin is required under these conditions; but an injection is equally inconvenient whether it contains fifty or sixty units and one has to weigh the discomfort of dietetic restrictions against the alleged wastage of a few units of insulin; furthermore, an increase in the carbohydrate content of the diet does not necessarily increase the insulin requirements. If the patient is allowed a free choice of diet, however, it is essential he be warned to take similar amounts of food at the same meals from day to day. Widdowson has shown that there are normally large daily variations in food intake, and it would be difficult to ensure proper stabilization under these conditions.

The Sort of Insulin. The insulins in common use can be characterized by their length of action. Soluble insulin begins to act in about thirty minutes and continues for about six hours; globin insulin takes slightly longer to begin acting and continues for approximately twenty-four hours; zinc protamine insulin goes on acting up to thirty-six hours. These differences are due to the various rates at which true insulin is released from the site of injection. It will be seen that an injection of soluble insulin should be followed by a meal containing carbohydrate and the general scheme is to have an injection of insulin morning and evening, each followed by a good meal. In this way most of the daily food intake occurs at breakfast and supper. With globin or zinc protamine insulin, on the other hand, it is necessary to spread the food as evenly as possible throughout the day. Thus, something should be taken at breakfast, between breakfast and dinner, at dinner, at tea, at supper, and before going to bed at night. If this is not inconvenient the advantage of only one injection per day points to the use of globin or zinc protamine insulin. Zinc protamine insulin at times raises red painful lumps at the sites of injection. This can sometimes be overcome by changing the make: but if not globin insulin should be used, since this rarely

gives rise to such difficulty. Patients who are unstable and therefore very difficult to control sometimes do well on two daily injections of globin insulin, one before breakfast and the other before supper.

Summary. The most important single aim in the treatment of diabetes is to keep the patient free from ketones. This can only be achieved by an adequate intake of carbohydrate, of which the metabolism is ensured by sufficient insulin. It may or may not be necessary to supplement the patient's own insulin by injections. The dose of insulin required is not necessarily correlated with the severity of the diabetes. For those patients requiring insulin the diet should be adequate for normal growth and activity and should be as nearly normal as possible, provided similar amounts are taken at the same meals, day by day, and a good intake of first-class protein and vitamin B complex is ensured.

Care of the feet is extremely important for all diabetics over forty.

The choice of the type of insulin for ordinary everyday use depends largely upon the daily feeding pattern of the patient.

REFERENCES

[1] G. A. Smart and C. H. Darling, *Lancet* 1948, ii, 630.

[2] R. W. Schnieder, Lena A. Lewis and E. Perry McCullagh, *Amercan Journal Med. Science*, 1946, ccxii, 462.

[3] R. D. Lawrence, *Physician's Bulletin*, 1947, xii, 34.

[4] V. P. Sydenstricker, L. E. Geeslin and J. W. Weaver, *J.A.M.A.*, 1939, cxiii, 2137.

I am indebted to the Bristol Royal Hospital for permission to use the illustration on p. 77.—G. A. S.

BLUE SCLEROTICS, FRAGILITAS OSSIUM AND DEAFNESS: REPORT OF A FAMILY*

BY

E. WATSON-WILLIAMS, *M.C.*, M.D., Ch.M.,

THAT a young and apparently healthy person should frequently break his bones, often from some trivial cause, seems bound to attract attention: still more, when this remarkable condition afflicts several members of one family, which is further distinguished by a startling blueness of what in others is "the white of the eye". Yet, although the condition is by no means rare, the first recognizable account appeared in 1788, when Eckman[1] described a family, three generations of which suffered from multiple fractures. Nearly a century elapsed before similar accounts were published in this country.[2]

In none of the early accounts is there any mention of the blueness of the sclerotic. Though of itself a striking phenomenon, it appears to have received no attention until 1896, when Spurway mentioned it casually at the end of an article on "Hereditary Tendency to Fracture."[3] The association of the two conditions was expounded by Eddowes in 1900.[4] Soon after this there appeared pedigrees of many families showing these two abnormalities:[5] in several of them, one or two individuals are recorded as being deaf. The importance of deafness was first shown by Bronson in 1917[6], who published two family trees: in one (Currie), among fifty-five persons, twenty suffered from fractures and had blue sclerotics, and seven of those were deaf.

In 1928 Bell[7] collected seventy-four pedigrees, with photographs, X-ray and colour drawings, illustrating the characteristic deformities. All the bones tend to be affected, the long bones especially. These

* Four members of the family were shown at the combined Summer Meeting of the sections of Laryngology and of Otology of the Royal Society of Medicine at Bristol on July 2nd, 1948.

I wish to acknowledge the help of Mr. G. F. Home, the Librarian of the Society, in the preparation of this article.—E. W.-W.

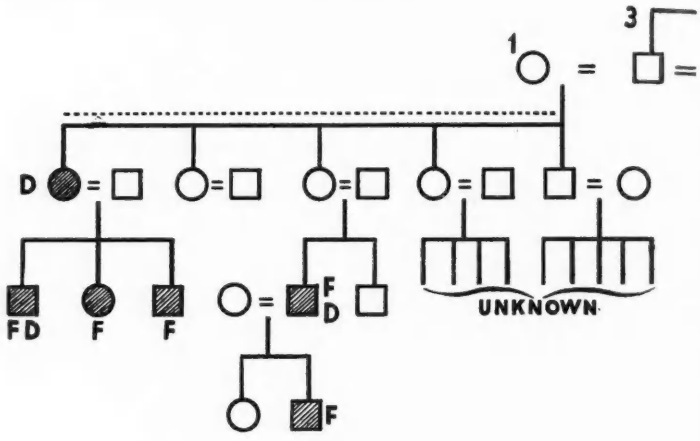
are thin, lacking density, and show typical cross-striation in X-ray films; they are easily broken—over a hundred fractures have been recorded in one patient—and there may be little pain or swelling accompanying the fracture. Repair is normal. Bending of bones is common, with resulting deformities of limbs, spine, pelvis, etc. The patients are usually of small stature, with "triangular" skulls produced by occipital and temporal (sometimes frontal) protuberances. In addition there may be abnormal laxity of ligaments: sprains and dislocations are frequent. Sometimes the tendency to fracture appears especially in one bone or limb. Fractures may occur before or during birth: the tendency to fracture diminishes in adult life.

The blueness of the sclerotics is due to these structures being abnormally thin, or perhaps only abnormally translucent,[5,6] so that the colour of the choroid is transmitted. As a rule the eyes show no other defect, but colour-blindness[9] and conical cornea[10] have been recorded. The blue sclerotics are present in all the patients, and are transmitted as a Mendelian dominant: of those with blue sclerotics two-thirds show liability to fractures, two-thirds become deaf in early adult life and nearly half show both changes. Those with blue eyes may transmit to their offspring the tendency to fractures or to deafness, although themselves not suffering from either. The sexes are equally affected and equally transmit the defect. Apparently, however, the condition may skip a generation:[11] and isolated cases occur without discoverable affected relatives—Nager's case (below) was one such.

The deafness usually appears at about the age of twenty and resembles that of otosclerosis: Rinne's sign negative (B.C.>A.C.) and bone conduction increased. But the nerve deafness which is a late phenomenon in otosclerosis here often occurs quite early. Cleminson[7] was able to publish a section from the temporal bone of a patient under Professor Nager (Zürich). It shows bone changes of the same order as those seen in typical otosclerosis; but whereas in the latter disease there is usually only a single focus, beginning just in front of the oval window, in Nager's case there were several foci involving not only that site, but the round window, canals, and internal meatus also. Such changes would explain the differences in deafness between this condition and "ordinary" otosclerosis.

The pathology of this condition is obscure. The blood shows no material abnormality; the blood calcium level may be rather high.[11] The usual "explanation" is a hereditary defect in the tissues of mesoblastic origin. There is no evidence of any endocrine abnormality. No treatment is known which influences the course of the disease. These patients are not suitable for the operation of fenestration of the labyrinth.

FIGURE 1



1 1st Husband of 3.

2 2nd Husband of 3.

3 Grandmother.

4 Mother of 6, 7, 8.

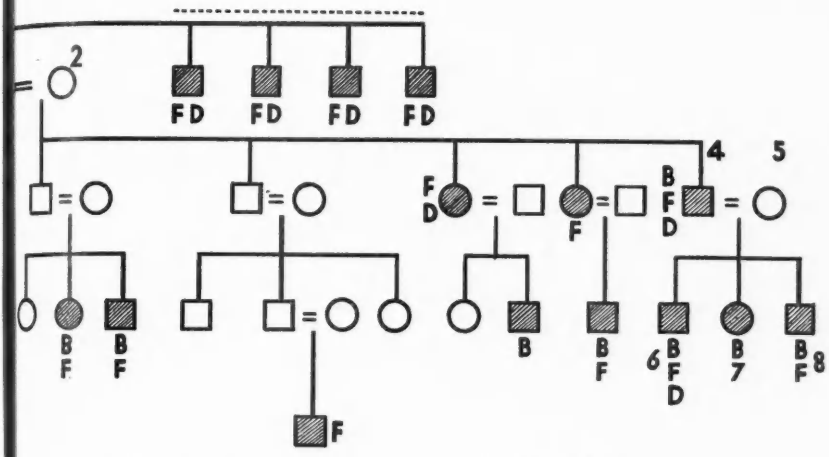
5 Father of 6, 7, 8.
His parents, brother and 5 sisters normal.

6, 7, 8 Three children.

The family, four members of which I bring to your notice, is interesting because the maternal grandmother married twice (Figure 1), and her descendants by both husbands show the triad under discussion. The grandmother herself, No. 3, has white sclerotics, has not broken her bones, and is not deaf : or rather, at seventy-two, has only recently begun to be deaf. But her four sisters all became deaf early in life and all suffered from fractures. (It has not been possible to discover the incidence of blue sclerotics among these, nor among descendants of the first husband ; we may reasonably assume that at least those who suffered from fractures or deafness showed eye changes also.) By her first husband, No. 1, she had four sons and one daughter. The eldest son and his three children are affected : the elder daughter of the third son, and *her* daughter, are also affected. Information is lacking about the other members of this family.

The second husband, No. 2, was normal : by him she had three daughters and two sons. The youngest daughter is No. 4, the

FIGURE 1



○ Normal male.	B Blue Sclerotics.
□ Normal female.	F Fractures.
◒ Affected male.	D Deafness.
◪ Affected female.	

Incidence of blue sclerotics unknown.

mother of the three children shown. Several of *her* nephews and nieces (on her own side of the family) and her grandniece, as well as her two brothers, are known to be affected. No. 5, the husband of No. 4, is normal, as are his brother and five sisters; I have not been able to find out anything about their descendants.

Gwyneth, No. 6, aged nineteen, came to me recently because she has begun to be deaf in the left ear during one year. She knows no cause for this, but "it runs in the family". She is a slim, dark, short girl (4 ft. 8 in.) with triangular head and bright blue sclerotics. She has broken her bones six times (left leg three times): X-rays show typical changes throughout the skeleton, but no abnormal deposit of callus. The audiogram (Table 2) shows loss of hearing in both ears, more in the left, by air conduction; the loss is greater for the high frequencies. (In typical otosclerosis the higher frequencies tend to escape, at least at first.) The bone conduction is normal. No tinnitus, membranes appear normal; never any earache or discharge.

TABLE 2

DECIBEL LOSS AT FREQUENCIES SHOWN

	CASE 6				CASE 4			
	R. ear		*L. ear*		*R. ear*		*L. ear*	
	AC	BC	AC	BC	AC	BC	AC	BC
128 ..	20		20		?	60	?	60
256 ..	10	*normal*	30	*normal*	?	60	?	70
512 ..	20		30		?	50	?	60
1,024 ..	10		30		?	50	?	50
2,048 ..	20		40			70		
4,096 ..			60					

Her mother, No. 4, became deaf at nineteen and now at forty-one has been very deaf for many years. Never any pain or discharge in either ear. The audiogram shows severe *nerve* deafness in both ears. She is a short, small woman with triangular head, marked scoliosis, and blue sclerotics ; as a child she had several fractures.

William, No. 7, aged ten, shows blue sclerotics : he is as tall as his elder sister, has never had a fracture, and his hearing is normal (tuning-fork tests only, 256 d.v., 2048, 4096).

Pauline, No. 8, aged 3, is a small child for her age, has blue sclerotics and bitemporal bulging of the skull. She has broken her left ankle, but her hearing is apparently normal.

REFERENCES

[1] Eckman, O. J., *Descriptio . . . osteom alaciae*. Upsala, 1788.

[2] Jones, J., *B.M.J.*, 1870, i, 86. Greenish, R. W., ib., 1880, i, 966. Pritchard, O., *Lancet*, 1883, ii, 394.

[3] Spurway, J., *B.M.J.*, 1896, ii, 844.

[4] Eddowes, A., ib., 1900, ii, 222.

[5] *e.g.* Harman, B., *Ophthalmoscope*, 8, 1910, 559.

[6] Bronson, E., *Edin. M.J.*, XVIII, 1917, 240. Fraser, J. S., *Proc. R. Soc. Med.*, Sect. Otol., XII, 1919, 126.

[7] Cleminson, F. J., ib., XX, 1927, 471.

[8] Bell, J., *Treasury of Human Inheritance*, London, 1928, Vol. II, Part 3, 269.

[9] Riddell, W. B., *Annals of Eugenics*, 10, 1940, 1.

[10] Behr, *Klin. Monatsbl. f. Augenh*, XVI, 1913, 281.

[11] Kersley, G. D., *St. Bart. Hosp. Rep.*, LXVIII, 1935, 159.

THIOURACIL IN THYROTOXICOSIS

A summary of the main points of Professor Himsworth's opening address in the Section of Medicine at the Annual Meeting of the British Medical Association at Cambridge, June 30th, 1948. With acknowledgments to the *British Medical Journal*, 1948, ii, p. 61.

It is now established that thiouracil can control the manifestations of thyrotoxicosis : patients vary widely in the speed of their response, but the majority are controlled within six weeks. No case is too severe for thiouracil treatment : to this may be attributed the virtual disappearance of the highly fatal thyroid crisis. A few cases react very slowly; those with nodular goitres and those previously treated with iodine. Omit iodine for a month before giving thiouracil : once thiouracil control is established iodine may be given.

Dosage.—Unnecessarily large doses have been used : a good response is obtained from 200 mgm. methyl thiouracil daily. Once symptoms are controlled, 25, 50—at most 100 mgm.—is sufficient ; more may produce myxoedema.

Exophthalmos (sclera showing below cornea), is found in only one-third of those with thyrotoxicosis and diffuse goitre, and in less than one-tenth of those with nodular goitre : in the majority the *appearance* of exophthalmos is due to lid retraction (sclera showing only above cornea). Thiouracil does not reduce true exophthalmos, but usually improves lid retraction.

Goitre.—The goitre may enlarge during treatment (over-dosage): some diminution in size is usual, but disappearance is rare. Nodular goitres remain unchanged.

Fibrillation.—Intermittent arrhythmia usually disappears : persistent arrhythmia is abolished by quinide after the thyrotoxicosis is controlled by thiouracil.

Cure.—Treatment must be continued for months after control is established : in patients who have had two years' remission after cessation of treatment, this had lasted 9–21 months ; of patients treated thirty months, two-thirds remain free from symptoms after cessation, and all in this group are well and at work.

Dangers.—In many patients the leucocyte-count drops during the first week of treatment : this can be ignored provided the total is not below 3000 with 50 per cent. polymorphs. About 10 per cent. of patients show idiosyncrasy, between sixth day and sixth week—

drug fever, rashes : continue treatment unless the polymorph count falls below danger point. Adenitis, lymphatic, salivary, etc., may be ignored. Agranulocytosis was seen in two per cent. of all cases treated : of these, one-quarter died. The lower doses now used and penicillin should reduce mortality : the polymorphs reappear within ten days of stopping thiouracil. Patients must be warned to stop the drug at once if they get fever or sore throat, and to call the doctor.

Surgery or Thiouracil.—Even the early figures given above show a mortality considerably less than that of the best surgeons : and included cases too bad for surgery. Thiouracil will do anything that surgery can : it is too early to judge permanence of results, but about one-third of the patients relapse sooner or later after the drug is stopped—another course may be given or the patient may prefer surgery.

Reviews of Books

Emergency Surgery : Part I. By HAMILTON BAILEY, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E. Sixth Edition. Illustrated. Pp. viii, 180. Bristol : John Wright & Sons Ltd. 1948. Price 21s. net.—Hamilton Bailey's *Emergency Surgery* (Part I) contains all the up-to-date information on the subject. It is rare to find an author with so fervent and trenchant a way of telling the surgical story. The newer procedures for transfusion of blood, etc., and the dangers from their abuse by the unwary are fully described and the simple safe Baxter type of infusion depicted. Anaesthesia, its risks and their remedies, are handled comprehensively. Abdominal trauma, appendicitis and other hypogastric sources of peritonitis are all well explained and afford a copiously illustrated guide for the younger surgeons. It is essentially a practical text-book and describes with meticulous detail the latest technical manoeuvres and explains their rationale. The author's style is appropriately incisive and forthright throughout, and it is obvious that he writes from the daily experience of one who is constantly at the "Surgical Bench". While nothing can replace the influence of experience in emergency surgery, it is from guides such as this that the young surgeon is inspired to aim at the highest standard of service.

Emergency Surgery: Part II. By HAMILTON BAILEY, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E. Sixth Edition. Pp. 181-388. Illustrated. Bristol : John Wright & Sons Ltd. 1948. Price 21s.—The first subject in this volume is fittingly one of the most beneficent chapters in emergency surgery—the closure of perforated ulcers—with

its sections on first-aid, mortality charts, new devices for dealing with difficult cases. One can want no better exposition on this catastrophe commonly overtaking busy men in the prime of life. All Britain needs is beds and doctors to give effect to such first-class instruction. In the biliary sphere, immediate operation might be emphasized in cases where the clinician senses virulent trouble but finds no decisive signs regarding pathology. Intervention in the incipient stage alone may disclose dire alternative diagnoses, such as sub-hepatic appendix, perforated ulcer or bowel. Operating promptly may therefore make all the difference, and need do no harm in the ordinary biliary case. The rarer casualties —swallowed foreign bodies and trauma—are handled in a lucid and competent manner. The page on obstruction after acute appendicitis is a model of practical wisdom in a most anxious situation. The patient, often youthful, a week or so later is suddenly at death's door. It is a tense situation from which he can only emerge safely and the initial surgical triumph be consummated by prompt second operation. Dealing with bowel obstruction due to gallstone, digital disintegration receives no mention. Admirable pictures and diagrams clarify the chapter on strangulated hernia. Dogmatic generalizations appear to which exception might be taken, and the page of sketches on biliary surgery is more picturesque than practical, but this liberally-illustrated book is a shining spearhead in the vanguard of modern surgery.

The Care of Tuberculosis in the Home. By JAMES MAXWELL, M.D., F.R.C.P. Second Edition. Pp. xii, 113. Illustrated. London: Hodder & Stoughton. 1947. Price 7s. 6d.—At once there looms before the individual confronted with a diagnosis of pulmonary tuberculosis a broad vista of urgent problems, both for himself and his family. If he is to gain the maximum benefit in health, either in his home or in sanatorium, the most vital problem is to make him acquainted, as rapidly as possible, with the nature of the disease, the treatment required, the dangers of infection and the necessary modifications of his future life. This book of 112 pages is written for the patient with this object in view, and it can be stated that the object is successfully achieved. Technical language has been avoided; the book is eminently readable and understandable. Some adverse criticisms can, of course, be found—for example, diagrams should be added; in discussing contacts the importance of X-rays could have been stressed and kissing deprecated. Umckaloabo is unworthy of a whole page, while streptomycin, para-aminosalicylic acid and B.C.G. vaccine receive no mention—and patients like to be up to date! Drawbacks to thoracoplasty are overrated, and the "acquisition of tuberculosis infection by the great majority of children within the first five years of life" is a highly questionable statement. It should be stated that milk is rendered safe by boiling. Resettlement and home crafts would have been welcomed. The price, 7s. 6d., may tend to limit the widest distribution which this book deserves.

Social Services in Bristol 1948. Compiled by the BRISTOL COUNCIL OF SOCIAL SERVICE. Pp. xx, 96. Bristol: J. W. Arrowsmith, Ltd. 1948. Price 3s.—This little book provides in convenient form a complete

guide to the city's social services. Beginning with a list of reference books, national and local, it continues with a summary of statutory services, and addresses of Government Departments, arranged in the same way. The largest part of the book is devoted to an alphabetical list of voluntary agencies, their objects, activities, and addresses. Another large section gives appendices on hospitals, homes, almshouses, clubs, and various societies. There are a very complete index and notes on " what to do in urgent cases". It is quite indispensable for all those active or interested in the social work of the city.

Meetings of Societies

Bristol Medico-Chirurgical Society

Programme for 1948-49

The provisional programme for the next session is as follows:

October 13th.—Annual General Meeting. Professor H. J. Drew Smythe's Presidential Address.

November.—**" Early Life "**, Professor A. V. Neale.

December.—Clinical Meeting.

January.—Professor Milnes Walker.

February.—Mr. V. B. Green Armytage (London).

March.—**" Chemotherapy "**, Dr. J. A. Boycott (Taunton).

April.—**" Coronary Thrombosis "**, Dr. G. E. F. Sutton; **" Renal Plication "**, Mr. A. V. Adams.

May.—**" Thymectomy for Myastheina Gravis "**, Mr. J. Keynes (London).

Royal Society of Medicine.

COMBINED MEETING OF SECTIONS OF LARYNGOLOGY AND OTOLOGY
BRISTOL, JULY 2ND AND 3RD, 1948.

ON July 2nd, Dr. A. W. Proetz (St. Louis, U.S.A.), read a paper on the physiology of the nose, with particular emphasis on the importance of preserving ciliary function. Recent work which he described adds further evidence to support views on this subject long held by British laryngologists.

Professor J. M. Yoffey described experiments in the investigation of communications between the meningeal spaces and the nasal mucosa. Dyes introduced into the nose, indian ink, and some virus proteins are

absorbed into the lymphatics, but do not enter the cranial cavity : but indian ink can pass in the reverse direction. Professor Yoffey remarked that our predecessors wrote of " purging rheum from the brain " by way of the nose. There may be new support for the accuracy of their views. In the afternoon a clinical meeting was held at the General Hospital : over one hundred members attended in addition to visitors. The main business of Saturday morning was a paper from Dr. F. Schiller (Oxford) on "**Purulent Pachymeningitis**" and the results of treatment by penicillin, combined with surgery. Very large doses of penicillin are required, and the results are improving. The lecturer raised the question whether prophylactic penicillin should become a standard procedure before operation on any of the nasal sinuses.

The South Western Laryngological Association.

CLINICAL meetings have been held at Bristol (October 18th, 1947), Bath (January 3rd, 1948), and Torquay (April 3rd). A large number of cases has been presented at each meeting, attendances have been good and the discussions of great interest and value. Mr. Watson Williams has been re-elected to the chair for 1948–49. The next meeting will be at Newport at 2 p.m. on January 8th, 1949.

The Society of Anaesthetists of the South-western Region.

A MOST successful meeting of the Society was held at Torquay on the 25th and 26th June, 1948. Demonstrations were given of the Etherinton Wilson spinal techniques for high and low anaesthesia, of Curare for endoscopy, and of Myanesin. During the meeting time was allowed for discussions on these techniques. Mr. R. H. Belsey, F.R.C.S., was the guest, and he gave a stimulating and provocative address entitled "**Some Problems of Thoracic Anaesthesia as seen by the Surgeon**". This was followed by open discussion and Mr. Belsey replied to the many questions. The next meeting of the Society will be held at Bristol on the 29th and 30th October, 1948. Dr. John Gillies, President of the Association of Anaesthetists of Great Britain and Ireland, will be the guest. Anaesthetists from other regions will be welcome and details may be obtained from the Hon. Secretary, Dr. G. L. Feneley, Avon Lodge, 8 Parry's Lane, Bristol 9.

British Medical Association, Bath Division.

On 16th July, 1948, at the Royal United Hospital, Bath, Professor Astwood of Boston, U.S.A., delivered a lecture on thiouracil in the treatment of thyrotoxicosis. After a survey of his early experimental work

he described the results obtained with thiouracil itself, and also with various related preparations and suggested that methyl thiouracil was one of the most satisfactory which he and his assistants had used.

He stressed the lengthy period during which treatment should be maintained, pointing out that to get the best results treatment must be pushed to the length of securing a mild degree of myxoedema. This stage he thought was better assessed by the clinical picture than by estimations of basal metabolic rate.

Professor Astwood indicated that the dangers of leucopenia and agranulocytosis were less serious than had been originally thought. He quoted figures from the Lahey Clinic (in which treatment was almost exclusively surgical), in support of his view that the results of thiouracil treatment compared favourably with those in surgical clinics of the highest standing.

TORQUAY AND DISTRICT MEDICAL SOCIETY

CLINICAL meetings have been arranged for November 25th, January 13th, February 24th and April 28th. Other fixtures are:

Nov.	11th.	Prof. C. Moir : **"Common Obstetrical Difficulties."**
Dec.	9th.	Dr. Kersley : **"Arthritis."**
Jan.	27th.	Mr. M. F. Nicholls : **"Retention of Urine."**
Feb.	10th.	Prof. A. Moncrieff : **"Diseases of the New-born."**
March	24th.	Mr. Belsey : **"Diseases of the Oesophagus."**
April	14th.	Dr. Brooks : **"Chest Disorders."**

CARDIFF MEDICAL SOCIETY

Programme:

Nov.	11th.	Dr. Mary Crosse : **"The Premature Baby."**
Dec.	9th.	Mr. Dickson Wright.
March	9th.	Sir Harold Gillies. (Annual Dinner.)
May meeting.		Dr. Geoffrey Hodgson : **"Dermatology."**

January, February and April meetings to be arranged later.

WESSEX RAHERE CLUB

THIS club held its first annual dinner at the Royal Hotel, Bristol on October 17th, 1948. Dr. E. R. Cullinan was Guest of Honour and some thirty-six members were present. Membership is open to all Bart's men resident in Somerset, Wiltshire and Gloucestershire. Mr. C. E. Kindersley (Bath) was elected Chairman and Mr. A. Daunt Bate-men (3, Circus, Bath) Hon. Secretary for the coming year. Dr. Cullinan gave an amusing discourse on "Present-day Bart's from within". It is hoped that any Bart's men who are interested, and have not yet been in touch with the club, will notify their whereabouts to the Hon. Secretary so that they can be kept informed of future meetings.

The Medical Library of the University of Bristol

WITH WHICH IS INCORPORATED

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the list in the Last issue.

American College of Surgeons (1)	1 volume.
Buenos Aires University (2)	1 volume.
Calcutta University (3)	2 volumes.
E. Wilfred Fish, Esq. (4)	1 volume.
Iowa State Medical Society (5)	3 volumes.
Michell Clarke Fund (6)	2 volumes.
Mrs. C. A. Moore (7)	22 volumes.
J. H. Perry, Esq. (8)	1 volume.
Public Health Institute, Chicago (9)	1 volume.
Dr. J. Samuels (10)	1 volume.
J. E. Shaw Fund (11)	1 volume.
Wilmer Ophthalmological Institute (12)	1 volume.
Messrs John Wright & Sons, Ltd. (13)	3 volumes.

Unbound periodicals have also been received from the "Black Bag", Dr. C. D. Evans, G. M. FitzGibbon, Esq., Professor R. Milnes Walker and Messrs. John Wright & Sons, Ltd.

THE ONE HUNDRED AND EIGHTY-NINTH LIST OF BOOKS.

The figures in round brackets refer to the figures after the names of the donors. The books to which no such figures have been attached have either been bought from the Library Fund or received through the *Journal*.

Bailey, H. H. .. .. *Emergency Surgery, Part 2* .. .. .. 7th Ed. 1948

Bailey, H. H. and Love, M. *Short Practice of Surgery, Part I.* .. .. 8th Ed. 1948

Bell, E. T. .. .. .. *Renal Diseases* .. .. (6) 1946

Bourne, A. W. and Williams, L. H. *Recent Advances in Obstetrics and Gynaecology* 7th Ed. 1948

Bowden, A. C. .. .. *Essentials of Local Anaesthesia in Dentistry* (13) 1945

Brahmachari, Sir U. .. *Gleanings from my Researches* .. 2 Vols. (3) 1940–41

British Pharmacopoeia 1948

Bronner, M. .. .. .. *Dental Surgeon's Handbook* .. .. 2nd Ed. (13) 1948

Carling, Sir E. R. and Ross, J. P. (Eds.) *British Surgical Practice* Vol. 3 (11) 1948

Cunningham, D. J. .. *Textbook of Anatomy* .. .. .. 8th Ed. (6) 1947

Currie J. R. and Mearns, A. G. *Hygiene* .. .. 3rd Ed. 1948

Daniels, M. and others *Tuberculosis in Young Adults—Royal College of Physicians Prophit Tuberculosis Survey*, 1935–44 1948

Dorland, W. A. M. .. *American Illustrated Medical Dictionary* 21st Ed. 1948

Duran, C. M. .. .. *Las Ciencias Medicas en Guatemala* .. 2nd Ed. 1945

Fish, E. W. .. .. *Surgical Pathology of the Mouth* .. .. .. (4) 1948

Gaddum, J. H. .. .. *Pharmacology* .. .. 3rd Ed. 1948

Gray, H. .. .. .. *Anatomy—Descriptive and Applied* 24th Ed. (8) 1939

Hale-White, W. .. .. *Materia Medica, Pharmacy, Pharmacology and Therapeutics* .. .. 27th Ed. 1948

Handfield-Jones, R. M. and Porritt, A. E. *Essentials of Modern Surgery* 3rd Ed. 1948

Hewer, C. L. .. .. *Recent Advances in Anaesthesia and Analgesia* 6th Ed. 1948

Hirsekorn, H. .. .. *Denture Base Readjustment* .. .. (13) 1943

Jennings, W. A. and Russ, S. *Radon—its Technique and Use* 1948

Maxwell, J. .. .. .. *Care of Tuberculosis in the Home* .. .. 2nd Ed. 1948

Molina, J. .. .. .. *Cavidades para Incrustaciones de Resinas Acrilicas* (2) 1944

Ogilvie, W. H. .. .. *Forward Surgery in Modern War* .. .. .. (7) 1944

Paterson, R. .. .. *Treatment of Malignant Disease by Radium and X-rays* 1948

Public Health Institute, Chicago *How Laymen Cut Medical Costs* .. .. (9) 1948

Samuels, J. .. .. .. *Endogenous Endocrinotherapy including the Causal Cure of Cancer* .. .. (10) 1947

Smout, C. F. Z. and Jacoby, F. *Gynaecological and Obstetrical Anatomy* 2nd Ed. 1948

TRANSACTIONS, REPORTS, JOURNALS, ETC.

American College of Surgeons' Year Book, 1947–1949 .. .. (1) 1947

Collected Reprints of the Wilmer Ophthalmological Institute. Vol. 8, 1946–1947 .. .. (12) 1948

International Clinics, 3rd Series, Vol. 4; 4th Series, Vols. 3, 4 (5) 1894–1895

Kongresszentralblatt fur Innere Medizin. Vols. 40–65 1926–1932

Quarterly Cumulative Index Medicus. Vol. 41, Jan–June, 1947 1948

Surgery, Gynecology and Obstetrics. Index Vols. 1–40 (1905–1925) 1926

Vols. 41–80 (1925–1945) 1946

Publications Received

From Messrs. Angus & Robertson, Ltd., Sydney :

" Elective Alimentary Rest " and the Elimination of so-called " Paralytic Ileus " after Abdominal Operations. By V. J. Kinsella, M.B., Ch.M. (Syd.), F.R.C.S., F.R.A.C.S. Price 3s. 1948.

The Mechanism of Abdominal Pain. By V. J. Kinsella, M.B., Ch.M. (Syd.), F.R.C.S.(Eng.), F.R.A.C.S. Price 32s. 6d. 1948.

From Messrs. H. K. Lewis & Co. Ltd. :

Infra-red Irradiation. By William Beaumont, M.R.C.S.(Eng.), L.R.C.P.(Lond.) with a foreword by Lord Horder, G.C.V.O., M.D., F.R.C.P. Third Edition. Price 8s. 6d. 1948.

Radiotherapy and Cancer. By A. G. C. Taylor, M.R.C.S., L.R.C.P., D.R., F.F.R. and others. Price 7s. 6d. 1948.

Psychiatric Case-taking Cards. By A. Spencer Paterson. Second Edition. Price 9d. each. 1948.

From Dr. E. Henty Smalpage :

The Universal Law of Chemical Decomposition. By E. Henty Smalpage, M.B., Ch.M., F.R.C.S. 1948.

From Sylviro Publications, Ltd. :

Handbook of Medicine for Final Year Students. By G. F. Walker, M.D., M.R.C.P. Fourth Edition. Price 25s. 1948.

From Messrs. John Wright & Sons, Ltd. :

British Journal of Surgery. Vol. 36. No. 141. August 1948. Price 12s. 6d. (Annual Subscription 42s.). 1948.

The Clinical Apprentice—a Guide for Students of Medicine. By John M. Naish, M.D., M.R.C.P. and John Apley, M.D., M.R.C.P. Price 15s. 1948.

Malignant Disease and its Treatment by Radium. By Sir Stanford Cade, K.B.E., C.B., F.R.C.S. Second Edition. Vol. 1. Price 52s. 6d. 1948.

Tuberculosis in Childhood. By Dorothy Stopford Price, M.D. Second Edition. Price 25s. 1948.

Demonstrations of Physical Signs in Clinical Surgery. By Hamilton Bailey, F.R.C.S., F.A.C.S. Second Edition. Part 2. Price 8s. 6d. 1948.

Local Medical Notes

On July 15th, 1948, The Stonebridge Press, the new printing works of Messrs. John Wright & Sons, Ltd., was formally opened by the Lord Mayor of Bristol, assisted by the Sheriff: one of the chief speakers at the luncheon afterwards was Sir Cecil Wakeley, Editorial Secretary to the *British Journal of Surgery*, one of the Company's best-known publications. In his speech he reminded his hearers that the *British Journal of Surgery* held an unrivalled position amongst surgical periodicals throughout the world, and that there was accordingly a tremendous demand for it wherever the Art of Surgery was practised. He deplored the fact that in spite of its intrinsic humanitarian worth and also of its value as an export, the policy of the *Journal* was still hampered by paper restrictions.

Mr. H. Chitty, Sheriff of Bristol, said that the publications of the House of Wright were renowned throughout the medical world: and the Lord Mayor, referring to " the vivid and serious enterprise " of the firm, said that he wished it ever-increasing success in its essential task of supplying medical textbooks to all quarters of the globe. Warm tributes were paid to the many printers in Bristol and the West of England who had helped the Company to maintain the technical excellence of its books during the seven years since the destruction of their Bristol factory, and also to the many work-people who had maintained their connection with the Company throughout.

Professor R. H. Parry, M.D., F.R.C.P., has been elected President of the Society of Medical Officers of Health.

EXAMINATION RESULTS.

University of Bristol.—Students of the University have recently passed the following examinations:

M.B., Ch.B.—*Supplementary First Examination:* J. Laltoo, A. J. MacDonald, Catherine M. Stephen.

B.D.S.—*Supplementary First Examination:* P. Crane, T. F. Harris.

L.D.S.—*Supplementary First Examination:* C. G. Innocent, D. McCandlish, D. R. Salter.

L.D.S., R.C.S.—*Final Examination. Part II:* R. J. P. Cousins.

M.R.C.S., L.R.C.P.—*Final Examination:* *Maro A. Wells (2, 3, 4), Kathleen J. Harrison (2, 4), *Suzanne K. R. Clarke (2), *Elizabeth Preston-Thomas (2, 3, 4), Molly I. Govier (2), *A. J. Lee (2, 3), *A. H. Levy (2, 3), R. J. Carey (2), B. F. Vaughan (2), Margaret Ford (3, 4), Medora Storkey (1), P. C. Farrant (1), Patricia M. Gilbert (1), P. A. Normandale (1), J. P. Martin (1).

* Qualified. (1) Pathology. (2) Medicine. (3) Surgery. (4) Midwifery.